embodiments, the B3 cell population has a density between about 1.052 g/ml and about 1.063 g/ml.

[0010] In all embodiments, the enriched renal cell population may be non-autologous to the native kidney. In all embodiments, the enriched renal cell population may be autologous to the native kidney.

[0011] In all embodiments, the products include paracrine factors, endocrine factors, juxtacrine factors, RNA, vesicles, microvesicles, exosomes, and any combination thereof. In one other embodiment, the vesicles include one or more secreted products selected from the group consisting of paracrine factors, endocrine factors, juxtacrine factors, and RNA. In another embodiment, the products are secreted from a renal cell construct comprising an enriched renal cell population directly seeded on or in a scaffold.

[0012] In all embodiments, the scaffold may contain a biocompatible material. In all embodiments, the biocompatible material may be a hydrogel.

[0013] In one embodiment, the present invention provides methods of assessing whether a kidney disease (KD) patient is responsive to treatment with a therapeutic. The method may include the step of determining or detecting the amount of vesicles or their luminal contents in a test sample obtained from a KD patient treated with the therapeutic, as compared to or relative to the amount of vesicles in a control sample, wherein a higher or lower amount of vesicles or their luminal contents in the test sample as compared to the amount of vesicles or their luminal contents in the control sample is indicative of the treated patient's responsiveness to treatment with the therapeutic. The vesicles may be kidney derived vesicles. The test sample may contain urine. The vesicles may contain a biomarker, which may be miRNA. The therapeutic may contain an enriched population of renal cells.

BRIEF DESCRIPTION OF THE DRAWINGS

[0014] FIGS. **1**A-**1**B show enrichment of epo-producing cell fraction from freshly-dissociated kidney tissue using a multi-layered step gradient technique (FIG. **1**A—left panel) or a single-layer mixing gradient technique (FIG. **1**B—right panel). Both methods result in the partial depletion of non epo-producing cell components (predominantly tubular cells) from the epo band, which appears between 1.025 g/mL and 1.035 g/mL.

[0015] FIG. **2** shows step gradients of "normoxic" (21% oxygen) and "hypoxic" (2% oxygen) rodent cultures that were harvested separately and applied side-by-side to identical step gradients.

[0016] FIG. **3** shows step gradients of "normoxic" (21% oxygen) and "hypoxic" (2% oxygen) canine cultures that were harvested separately and applied side-by-side to identical step gradients.

[0017] FIG. **4** shows histopathologic features of the HK17 and HK19 samples.

[0018] FIG. **5** shows high content analysis (HCA) of albumin transport in human NKA cells defining regions of interest (ROI).

[0019] FIG. **6** shows quantitative comparison of albumin transport in NKA cells derived from non-CKD and CKD kidney.

[0020] FIG. **7** depicts comparative analysis of marker expression between tubular-enriched B2 and tubular cell-depleted B4 subfractions.

[0021] FIG. **8** depicts comparative functional analysis of albumin transport between tubular-enriched B2 and tubular cell-depleted B4 subfractions.

[0022] FIG. **9** shows expression of SOX2 mRNA in host tissue after treatment of 5/6 NX rats.

[0023] FIG. **10** Western blot showing time course of expression of CD24, CD133, UTF1, SOX2, NODAL and LEFTY.

[0024] FIG. **11** depicts a time course of regenerative response index (RRI).

[0025] FIG. **12** provides a schematic for the preparation and analysis of UNFX conditioned media.

[0026] FIGS. **13**A-**13**D show that conditioned media from UNFX cultures affects multiple cellular processes in vitro that are potentially associated with regenerative outcomes. FIG. **13**A shows that UNFX-conditioned media attenuates TNF-a mediated activation of NF-kB. FIG. **13**B shows that UNFX-conditioned media increases proangiogenic behavior of HUVEC cell cultures. FIG. **13**C shows that UNFX-conditioned media attenuates fibrosis pathways in epithelial cells. FIG. **13**D depicts the positive feedback loop established by TGFβ1 and Plasminogen Activator Inhibitor-1 (PAI-1).

[0027] FIGS. **14**A-**14**B show a Western blot analysis demonstrating the attenuation of fibrosis pathways in mesangial cells.

[0028] FIGS. **15**A-**15**C shows that the conditioned media from UNFX contains secreted vesicles. FIG. **15**A depicts secreted vesicles, which are bilipid structures (red) that encompass cytoplasm-derived internal components (green). FIGS. **15**B-**15**C show FACS sorting.

[0029] FIG. **16**A shows a Western blot in which total protein was prepared and assayed for PAI-1 and bActin. FIG. **16**B depicts the microRNA, miR-30b-5p.

[0030] FIGS. **17**A-**17**C show representative immunohistochemistry images of PAI-1 in Lewis rat kidneys following delivery of bioactive kidney cells after undergoing a nephrectomy. FIG. **17**D shows a comparison of PAI-1 expression in untreated, nephrectomized rats (red squares), treated, nephrectomized rats (blue diamonds), and control animals (green triangles). FIG. **17**E shows representative Western blot analysis on kidney samples taken at 3 and 6 months post-treatment. FIG. **17**F shows a 2-hour exposure to NKA conditioned media reduces nuclear localization of NFκB p65. FIG. **17**G depicts the canonical activation of the NFkB pathway by TNFα.

[0031] FIGS. **18**A-**18**B show the nuclear localization of NFkB p65 subunit in animals with (A) progressive CKD initiated by 5/6 nephrectomy and (B) non-progressive renal insufficiency initiated by unilateral nephrectomy. FIGS. **18**C-**18**D show (C) a Western blot analysis for NFkB p65 in extracts of Lewis rat kidney tissue that have undergone the 5/6 nephrectomy; and (D) electrophoretic mobility shift assay (EMSA) on extracts. FIG. **18**E shows immunohistochemical detection of the NFκB p65 subunit in tissue obtained from Lewis rats with established CKD that received intra-renal injection of NKA (panel A) or non-bioactive renal cells (panel B).

[0032] FIGS. **19**A-**19**C show in vivo evaluation of biomaterials at 1 week and 4 weeks post-implantation.

[0033] FIGS. **20**A-**20**D show live/dead staining of NKA constructs. FIGS. **20**E-**20**G show transcriptomic profiling of NKA constructs.